2. Sera from *recent, rapidly convalescent and nonparalytic patients* show high antiviral titers: these individuals are most prolific sources of carriers.

3. Human vectors harbor the virus in their intestinal and respiratory tracts.

4. Identification of virus, and hence detection of carriers, is by means of time-consuming, expensive and difficult biological tests, and is uncertain at present.

5. Proof of noninfectiousness ultimately must be the criterion for release from quarantine.

6. Studies in pathology show that (*a*) poliomyelitis is a systemic disease primarily, and a central nervous system disease secondarily; (*b*) the systemic phase usually precedes or accompanies the central nervous system phase, which may be absent altogether; (*c*) the nervous lesions are diffuse below the midbrain, in spite of apparent clinical localizations; (*d*) the degree of pathologic change in the central nervous system does not parallel the clinical picture; (*e*) the meningeal involvement is only commensurate with the degree of systemic infection, and may be absent entirely; (*f*) cellular changes in the spinal fluid are proportional to the degree of meningitis present; and (*g*) the spinal fluid cell count may remain entirely normal and unchanged.

7. Diagnosis in many instances does not depend on spinal fluid corroboration.

8. Recognition of the nonparalytic case is possible and most desirable, as this individual is a latent source of potential infection.

9. Present prophylactic vaccines offer encouragement, but no convincing proof of immunity, and Kolmer admits the morbidity is greater in the vaccinated group than in the unvaccinated population.

10. Immunotransfusion is recommended early in severe cases.

11. Convalescent serum by all routes has proved beneficial in our experience.

12. Hypertonic dextrose in normal saline intravenously has proved advantageous in the systemic phase of the disease, and Retan believes the hypotonic solution saves lives in the paralytic group when given by his method.

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POLIOMYELITIS: ITS TREATMENT*

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THE treatment of poliomyelitis is an ungrateful subject for discussion, for it has taken us many years to appreciate the importance of a few measures, proved useful, essential to the proper care of the patient. These measures are so few and so simple that a discussion of treatment should best reiterate and reëmphasize them. A vast amount of scientific study of this disease has been productive of a pathetic paucity of information regarding specific curative efforts which can serve as basis only for speculation as to the future of treatment.

We may properly divide our discussion into three phases: (1) the essentials of management of the typical attack apart from paralytic manifestations; (2) the treatment of the various forms of muscular

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weakness and their attendant complications; and (3) a brief consideration of the theory and application of specific therapy.

EARLY DIAGNOSIS IMPORTANT

Early diagnosis is an important function of proper treatment. Often this diagnosis is based on little more than a sound clinical impression which is induced, prior to the appearance of paralysis, by the summation of a number of factors which may include the prevalence of an epidemic, a history of exposure, and the presence of illness and prostration to a greatly varying degree, and accompanied by any or all of the systemic manifestations of an attack. Of prime importance in the examination of the patient are tremulousness, fatiguability and the presence of neck and back stiffness, which are usually not extremely marked and are limited to the last few degrees of flexion of the head on the chest. Examination of the spinal fluid necessitates a certain amount of trauma to the patient, but this may be minimized by skillful performance of puncture, and is justified by the fact that the evidence thus secured usually quickly establishes or disproves the diagnosis. It is quite rare for the spinal fluid to be normal in the presence of suspicious clinical symptoms in a case in which it is ever possible conclusively to establish the diagnosis—although exceptions to this rule do occur.

EARLY PROCEDURES IN TREATMENT

In the early stage of infection, when the available evidence supports the probability of the diagnosis, certain measures should always be promptly instituted. The patient should be carefully isolated for the protection of the community. The communicability is low; but the disease almost certainly spreads through contact, and it can hardly be doubted that contagiousness is greater at the onset than at any subsequent time. The patient should be placed under conditions which absolutely protect him from muscular and nervous fatigue; he should be made as comfortable as possible and should be subjected to the minimum of examination necessary to observe his progress. Fatigue is an important matter in increasing susceptibility to the disease, and its avoidance in the early stage of the infection best serves to cut down the extent of subsequent paralytic development. Whatever may be expected from specific therapy can best be secured only at this time.

As the disease progresses, the onset of paralysis may be most insidious, and is overlooked with surprising frequency. It is good practice to anticipate common forms of weakness by providing some support for the arms and feet. The arms may be supported on pillows in a position of abduction and slight external rotation, which spares the deltoids and the rotators of the arm. The feet may be supported with pillows or sand bags as partial protection against the insidious development of foot drop.

The patient should be handled in a gentle manner, and every effort made to dispel his fear—which is sometimes difficult—and to minimize his activity. General care is similar to that applied to the common, mild acute infections; diet and elimination present no great problem and, in these early preparalytic cases, restlessness and irritability are seldom difficult to control. All accounts of modern epidemics indicate that, with the application of only these simple measures, one need not expect a high incidence of paralytic or fatal sequelae, and that, as our efforts increase in detecting the disease early and treating it thus simply, we may expect the prognosis steadily to improve.

TREATMENT OF PARALYSIS

In the majority of cases typical weakness of skeletal muscles, if evident at all, appears on the third or fourth day of the fever, although occasionally it puts in its appearance after a prolonged febrile course. In only one form of the disease does paralysis characteristically come early, and this is the so-called bulbar (actually bulbo-pontine) form in which weakness of the muscles supplied by the cranial nerves may be detected almost at the onset of the fever. Involvement of the muscles of the eye, the face, of mastication, of swallowing and phonation, should always be sought for and promptly recognized. These signs indicate a form of the infection most immediately dangerous to life; but with the best prognosis for complete recovery if survival occurs. The diagnosis may frequently be made through the observation of a collection of saliva in the back of the throat, which indicates the inability of the patient to swallow. Serious progression may rapidly ensue, and a considerable proportion of poliomyelitis deaths are caused by central respiratory failure, which amounts to a complete disorganization of respiratory function. This central type of respiratory failure is amenable only to a very small extent to the use of the respirator; the patient breathes irregularly, adapts himself poorly to the rhythm of the machine, is unable to free his air passages from secretion; and aspiration pneumonia frequently complicates the picture. Respiratory stimulants are, naturally, of little avail. With evident involvement of swallowing and respiration, the patient should be placed on his face, the foot of the bed elevated, and suction should be used to clear the air passages. This patient should not be fed by gavage during the active course, but should be supported by the parenteral administration of fluids and dextrose. When failure of respiration impends, the respirator should be tried; it may even be justifiable to suppress the patient's incoördinated respiratory efforts, hiccoughs, etc., by the use of fairly large doses of morphin, while the use of the machine is being instituted. Usually, the course of cranial-nerve paralysis is short: death quickly ensues or the patient speedily recovers function to a considerable degree. When cranial nerve involvement is accompanied by skeletal involvement, the latter should also be appropriately dealt with.

Skeletal Muscle Weakness.—The appearance of skeletal muscle weakness should be carefully sought during the active stage of the disease. Adequate examinations can be conducted daily with a minimum of disturbance. The detection of actual muscle weakness is of far greater importance than meticulous examination of the reflexes. As soon as there is the slightest amount of muscle weakness the involved extremity should be supported at rest in a

position which protects the damaged function from the effects of gravity and the pull of opposing muscles. Temporarily, during the early stage, the arms may be supported by means of pillows or slings attached to the head of the bed. The legs may be temporarily supported by sand bags or pillows. The muscles of the trunk require only the recumbent posture during the early stage. It is of extreme importance that these measures of support be instituted at the earliest possible moment. Despite all that has been said about this matter, slight muscle weakness all too frequently goes unrecognized or persists untreated until the end of the quarantine period, with immeasurable harm to the ultimate prognosis. Make-shift methods, sand bags, pillows, and slings should quickly be superseded by methods of support of better design. Many of the orthopedists prefer lightweight splints because of the ease with which they can be applied or removed. My own preference is for very lightweight plaster casts, which can quickly be abbreviated to a half shell and, while lacking the advantage of easy application, have at the same time the advantage that they are not apt so frequently to be removed. These measures for support do not demand superior skill although, if the orthopedist is to assume the later care of the patient, he should properly be consulted at an early stage so that the patient may receive the advantage of continuity of treatment.

Pain.—Pain is a common concomitant of the paralytic stage and its relief is difficult. Barbiturates are not conspicuously successful and narcotics are to be avoided. The application of heat, in any manner, is productive of increased comfort, and radiant heat is especially helpful. Sedation is best secured by trial of a variety of agents, including hypnotics, sedatives, and narcotics; and paraldehyd given by mouth or by rectum is very helpful.

Respiratory Weakness.—Respiratory weakness is frequently referred to as bulbar paralysis, but this term is often erroneously applied, inasmuch as the common form of respiratory involvement concerns the innervation of the intercostal muscles and the diaphragm, and is less likely to be due to central involvement. When there is involvement of one or both shoulder girdles, respiratory weakness should expectantly be watched for. This may develop most insidiously; there is a gradual diminution of chest excursion and increasing loss of ability to maintain the expansion of the chest against the pull of the diaphragm. Evident respiratory distress is usually lacking, although the patient shows increasing anxiety; the increased activity of the diaphragm leads to progressive diaphragmatic fatigue which may terminate abruptly with cessation of breathing. These typical cases respond well to the action of the respirator; the patient should be placed in the machine early, before there is complete failure, to become, usually, immediately comfortable. The machine simulates closely the physiology of breathing, and acts to some extent like a splint in that it spares the damaged respiratory muscles overfatigue. The respirator can maintain function only as a temporary expedient until subsidence of active disease permits restoration of all or part of the damaged respiratory function, which occurs in a surprising proportion of cases. That function does not invariably return is no fault of the method; the minority of cases in which normal respiration is not resumed to some extent, and in which the respirator simply prolongs a miserable existence, is an unfortunate occurrence which is compensated for by many in which this function does return and the patient finally enjoys restoration to a useful existence. It is almost obligatory that the patient in the respirator be handled by those adequately skilled in its operation, and the coöperation of a skilled team should always be developed for this purpose. It is more than a trick to be able to place the patient in the machine deftly and to care for his wants, nutrition, prevention of decubitus, elimination, etc., while he is totally dependent on his attendants.

Bladder and Bowel Weakness.—Bladder and bowel weakness commonly accompany weakness of the muscles of the lower abdomen and back, and unless adequate precautions are taken to prevent it, difficult defecation will be complicated by the accumulation of masses of impacted feces which are difficult to remove. Catharsis is less helpful than the use of lubricants, supplemented by gentle flushing and enemas of the lower bowel. Bladder weakness, formerly believed to be unusual in poliomyelitis, is not uncommon. Opinion is divided whether these patients should be catheterized or permitted reflex emptying; but, despite the danger of bladder infection (which commonly supervenes), it is usually better, in my opinion, to employ catheterization to relieve distention. As a rule, bladder function quickly returns, usually within a week after defervescence.

COMMENT

All of the useful methods of the acute stage should be continuously employed, and three to six weeks after onset a slow return to activity and painstaking reëducation of damaged function should be started. This should be carried out under the best orthopedic and physiotherapeutic supervision obtainable, and should proceed on the basis of an accurate estimate of muscle function with an appropriately planned program. The final outcome is almost invariably better than the condition at the height of the disease, and slow improvement may be expected under proper care for one, two, or three years. Physiotherapy in the pool is of great advantage, despite the fact that it is popularly overrated. Its chief value is that of permitting muscular activity without weight bearing, thus facilitating reëducation of damaged, but not destroyed, function. There should be no hard and fast rule about the time for substitution of surgical procedures for conservative treatment; when weakness does not cause deformity and impairment of surviving function, conservative measures may be hopefully continued for a long time; but where persistent weakness leads to severe and incorrectible deformity, as in involvement of back and trunk muscles, it may be advisable to intervene surgically very early.

The proved essentials of the treatment of poliomyelitis depend on simple principles which should be recognized by all practitioners; their proper employment taxes the art of medicine to the extreme. Final prognosis is best served by an early

diagnosis, a persistent application of simple orthopedic procedures for protection against deformity and disability, and the patient persistence in re-education and rehabilitation of the patient, which must include an effort to maintain in every way his morale and coöperation.

SPECIFIC THERAPY

Very little need be said about specific therapy, which is highly controversial and mostly experimental in nature. There have been advocates of many forms of treatment, including x-ray, diathermy, intravenous injections of hypertonic solutions to reduce edema, forced spinal drainage, and various efforts at chemotherapy, including sulfanilamide and related drugs. So far all of these have failed to prove their value. The most controversial subject involves the use of convalescent serum, and immune or hyperimmune serum for early treatment.

The principle of treating poliomyelitis with human immune serum rests on the evidence that the serum of many convalescents, as well as many who have no history of the disease, contains neutralizing antibodies for the virus. Opinions regarding the potential value of this theory of treatment are hopelessly conflicting, but many of the contrary opinions have been voiced by such ex cathedra authority that their views are very commonly accepted. I would not, for a moment, support the contention that the value of serum treatment has been absolutely proved, but I am strongly of the opinion that it is a promising method which has not been proved valueless by all the evidence submitted against it.

It is perfectly true that most of the assembled large series of cases treated by serum have shown no statistical improvement in the end-results. According to Park,[1] Fischer,[2] and others, this is sufficient evidence that it is of no value. The cases which support this view almost exclusively include those treated with relatively small amounts of serum. This is an extremely critical test, for small amounts of serum could conceivably benefit the general run of cases only if the serum were tremendously potent. This disease is admittedly resistant to therapy, and serum is not hyperimmune, but only weakly antiviral in action. It could scarcely be anticipated that treatment of a resistant disease with a weak serum would give statistical evidence comparable to that of routine treatment of diphtheria with antitoxin. What statistical evidence could be provided, for example, by antitoxin treatment of diphtheria with a routine of 100 to 500 units as a standard dose, particularly if every third dose were entirely lacking in antitoxin?

Significant analogies are presented by other infections. Meningococcus disease treated with antimeningococcus serum does not show in statistical studies, from all over the country,[3] a great departure from the untreated mortality. Here we deal with virulent infection treated with a weakly antibacterial agent. Most clinicians of experience, nevertheless, grant that antimeningococcus serum demonstrates its value in many individual cases. A comparable state of affairs exists in measles: a recent paper showed convincingly that the course of measles is unaffected by 20 to 40 cubic centimeters of adult blood (weakly antiviral), given at the onset of symptoms.[4] Equally convincing reports show that intravenous injection of 50 to 200 cubic centimeters of convalescent serum (more strongly antiviral) will abort an attack even after onset.

Even though they are in the minority, excellent clinicians from all over the world have expressed the opinion that the course of poliomyelitis is beneficially affected by large amounts of convalescent serum (Jensen,[5] Harmon,[6] Levinson[7]). Best of all these is the experience of Levinson, whose series, treated with large doses, has some statistical merit. Opinions based on intensive treatment of a few cases may be in error, but should not be discredited on the basis of a series treated with homeopathic doses.

Convalescent serum does not greatly benefit experimental infection in monkeys after the onset of symptoms. This, too, is a most bitterly critical test under which many therapeutic methods would also fail. The production of infection in naturally insusceptible animals requires such large doses of virus as to be quantitatively beyond the possibility of treatment and totally incapable of comparison with the natural attack in susceptible human beings.

It has been said that virus infections gain entrance within the cells and are completely insusceptible to therapeutic serum. If this general principle is common to virus infections, it should apply to measles which, however, can be definitely aborted by large amounts of convalescent serum. We recently, and quite accidentally, had a similar experience in aborting chicken-pox by a transfusion given at the onset of the eruption. The virus of poliomyelitis in the nervous tissue may be insusceptible to serum therapy, but the only test of this hypothesis will be actual experience in poliomyelitis, and cannot rest on conjecture, however well founded.

It is commonly stated that the choroid barrier prevents antibodies in the circulation from reaching lesions in the central nervous system.[8] This rests on a poor conception of the physiology of the central nervous system. The choroid barrier lies between the blood stream and the spinal fluid, the principle might apply to meningitis (although it does not), but the spinal fluid is not the nutrient medium of the brain and cord, and antibodies can best be carried to the depths of the nervous system through the blood, without the necessity of first reaching the spinal fluid. It can quickly be demonstrated at any convenient bar that certain substances can quickly be brought to the brain cells by the blood, and it has recently been conclusively shown that antibodies can also thus quickly reach the brain cells.[9] Somewhat concerned with this theoretical question is the proposal that antibodies should be administered intrathecally, a form of treatment originally sponsored by some of those now bitterly opposed to any similar form of therapy. It seems altogether illogical that antibodies should be given into the subarachnoid space in order to reach the lesions of poliomyelitis; for there is, in this disease,

no true meningitis, the virus has not been found in the spinal fluid, antibodies reach the brain and cord less easily through the spinal fluid than through the blood, and it is beyond doubt that such a procedure causes discomfort and actual danger to the patient.

The status of treatment of poliomyelitis with an antiviral serum can fairly be summarized with the statement that none of the arguments against its potential or proved value successfully demolish its rationale, and the opinions of numerous observers support its use in clinical grounds as cogent as those applying to many measures in current use in other infections. The crux of the whole question is, after all, not so much whether serum has proved its value as whether the method of treatment has any *promise* of value. Those who are interested in this form of therapy should continue entirely on an experimental basis, using very large doses of serum. This may not be practical routine, but is essential to the final status of this approach to therapy.

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DIAPHRAGMATIC HERNIA: RESULTS OF SURGICAL TREATMENT IN 210 CASES*

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II †

SURGICAL TREATMENT

DIAPHRAGMATIC hernia is primarily a mechanical condition, and the only treatment which will relieve the condition is operative repair or reconstruction of the abnormal opening in the diaphragm. The indications for surgical intervention and the methods and technique of surgical procedures depend on the type, situation, and size of the defect in the structure of the diaphragmatic muscle, the kind and amount of abdominal viscera involved in the hernia, and whether or not the viscera are enclosed in the hernial sac. I shall first describe the general surgical methods and then consider the special technique which is required in the surgical treatment of some types of hernia.

From the standpoint of treatment cases of hiatus hernia may be divided into three groups: in the first group the hernia is small and is recognized roentgenologically, often during the course of a general examination, and causes few or no clinical symptoms. No treatment is indicated in this group of cases. The second group includes those cases in which the symptoms are moderate and the hernias are of moderate size; in many of the cases in this group, conservative treatment, such as regulation of diet and reduction of weight, is sufficient to relieve the symptoms. The third group includes those cases in which there is no response to conservative measures; in these cases the hernias usually are large, and in many cases, in my experience, there are complications, such as incarceration of the stomach or gastric erosion. In this group of cases the only treatment that assures relief of symptoms is operative repair of the hernia.

In all cases in which a third or more of the stomach is involved in the hernia, surgical intervention should be considered because the condition is progressive and usually becomes rapidly worse after the hernia has attained this size. Operation should be performed before severe incarceration, with consequent obstruction and traumatic lesions of the stomach, has occurred. The operative risk is increased by gastric retention, and the technical difficulties are enhanced by fixation of the stomach to the diaphragm and to the hernial sac within the thorax. In all cases in which the colon is involved in the hernia, early operation is necessary because of the danger of intestinal obstruction.

Other types of hernia, such as traumatic hernia or those in which there is a congenital absence of a portion of the diaphragm, should be treated surgically, because the colon and small bowel are usually involved in the hernia and there is great danger of intestinal obstruction. In cases of traumatic hernia it is best not to operate until the acute symptoms caused by the primary injury have subsided, if the patient's condition will permit this delay.

Interruption of the Phrenic Nerve.—Paralysis of the diaphragm, produced either by temporary or permanent interruption of the phrenic nerve, is of value as a procedure preliminary to radical operative repair of many different types of diaphragmatic hernia. It is a necessary procedure in the surgical treatment of partial thoracic stomach resulting from a congenitally short esophagus. In some cases in which radical operative repair is contraindicated, it may be used as a palliative measure. In most instances in which interruption of the phrenic nerve is utilized as a procedure preliminary to radical operative repair of the hernia, I prefer, first, to perform temporary interruption of the nerve by crushing it, because in many instances it may not be necessary for the paralysis to be permanent. Function is usually reëstablished in three to six months. In cases in which reestablishment of function of the diaphragm is not desirable because of the danger of recurrence of the hernia, the paralysis can be made permanent by cutting or avulsing the phrenic nerve. As a procedure preliminary to radical surgical treatment,

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